

# Acute whole-body vibration increases reciprocal inhibition

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## ABSTRACT

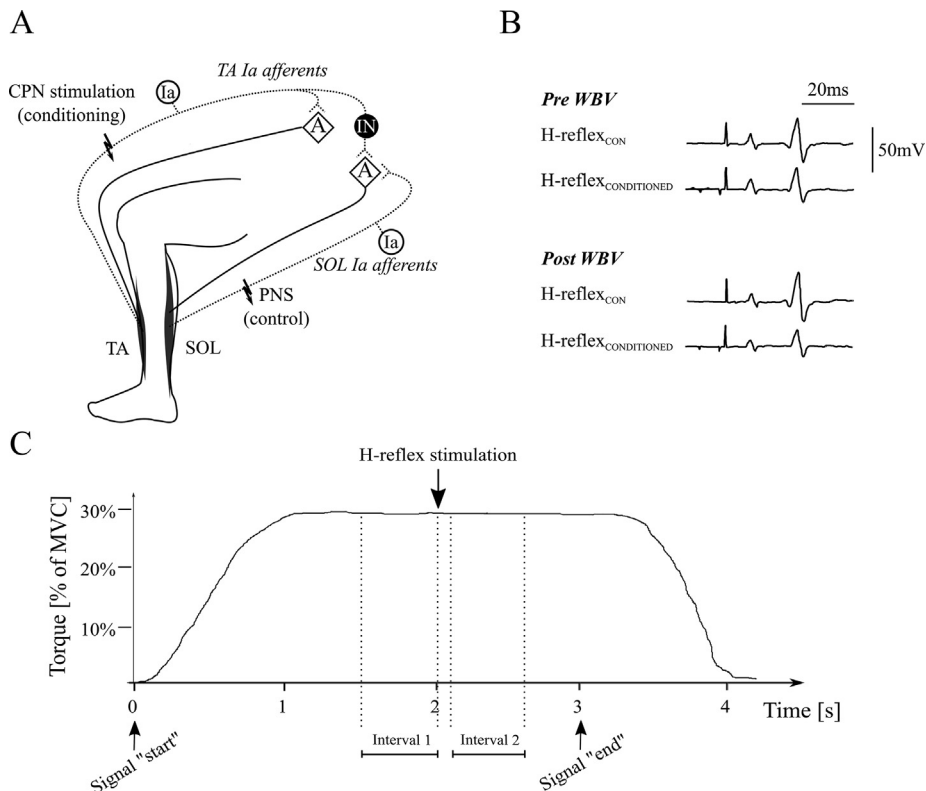
Based on previous evidence that whole-body vibration (WBV) affects pathways involved in disynaptic reciprocal inhibition (*DRI*), the present hypothesis-driven experiment aimed to assess the acute effects of WBV on *DRI* and co-contraction. *DRI* from ankle dorsiflexors to plantar flexors was investigated during submaximal dorsiflexion before and after 1 min of WBV. With electromyography, musculus soleus (SOL) H-reflex depression following a conditioning stimulation of the peroneal nerve (1.1x motor threshold for the musculus tibialis anterior, TA) was assessed and co-contraction was calculated. After WBV, *DRI* was significantly increased (+4%,  $p < 0.05$ ). SOL (−13%,  $p < 0.05$ ) and TA (−6%,  $p < 0.05$ ) activities were significantly reduced; co-contraction tended to be diminished (−8%,  $p = 0.05$ ). Dorsiflexion torque remained unchanged. After WBV, *DRI* increased during submaximal isometric contraction in healthy subjects. The simultaneous SOL relaxation and TA contraction indicate that a more economic movement execution is of functional significance for WBV application in clinical and athletic treatment.

## 1. Introduction

In the last decade, increasing emphasis has been placed on neuromuscular training to improve motor control during human movement. In this context, whole-body vibration (WBV), the use of high-frequency mechanical oscillations to stimulate skeletal muscles, has been brought to the forefront (Rittweger, 2010). Numerous studies have demonstrated improved performance in response to WBV, including increases in strength (Delecluse, Roelants, & Verschueren, 2003; Roelants, Delecluse, & Verschueren, 2004; Torvinen et al., 2002), power (Rees, Murphy, & Watsford, 2008; Roelants et al., 2004), and rate of force development (Cochrane, Stannard, Firth, & Rittweger, 2010) in isometric and dynamic muscle actions. Furthermore, experiments have shown that WBV acts on neuromuscular coupling and improves motor coordination (Cochrane, 2010; Ness & Field-Fote, 2009; Stark, Nikopoulou-Smyrni, Stabrey, Semler, & Schoenau, 2010). Although it has been argued that neural enhancement at the spinal level may underlie such WBV-induced improvements, the mechanisms and neuromuscular potentiation effects have received little attention (Cochrane, 2011).

Neurophysiological research has highlighted that accurate and effective movement execution requires remarkably precise coordination of the involved agonistic and antagonistic muscles. Neuronal circuitries in the spinal cord are pivotal to ensuring synergistic and antagonistic muscle coordination (Nielsen, 2004). One well-established mechanism involves the disynaptic reciprocal inhibitory (*DRI*, Fig. 1A) pathway (Crone, Nielsen, Petersen, Ballegaard, & Hultborn, 1994). Reciprocal inhibition is defined as the inhibition of antagonistic alpha motor neurons evoked through contraction of the agonistic muscle (Crone, 1993) under the control of supraspinal centers (Pierrot-Desseilligny & Burke, 2012). The Ia muscle spindle afferents innervate the homonymous alpha motor neuron, which causes the muscle to contract (Crone, 1993). Simultaneously, an inhibitory interneuron is innervated at the alpha motor neuron, which synapses onto the antagonistic muscles (Pierrot-Desseilligny & Burke, 2012). The activation of this inhibitory

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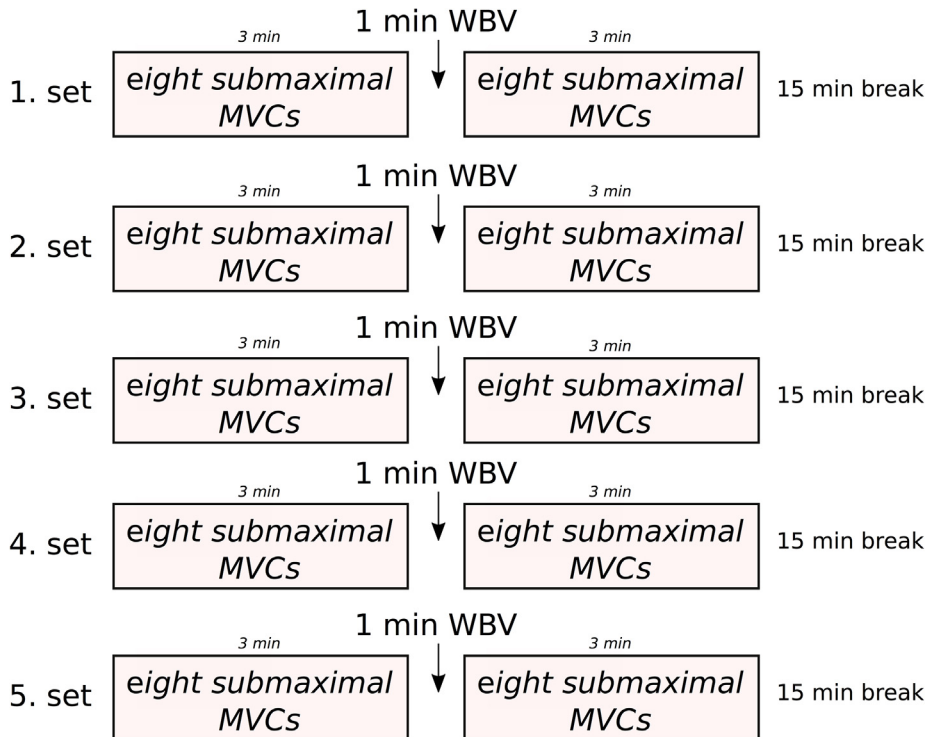
**Fig. 1. Study methodology and setting.** **A** illustrates that disinaptic reciprocal inhibition was assessed by peripheral nerve (PNS) and common peroneal nerve (CPN) stimulation at different conditioning-test intervals, leading to a conditioning of the H-reflex. **B** shows an example of an unconditioned ( $H\text{-reflex}_{\text{CON}}$ ) and conditioned ( $H\text{-reflex}_{\text{CONDITIONED}}$ ) H-reflex during submaximal dorsiflexion before and after WBV. **C** illustrates a submaximal dorsiflexion torque of a representative subject. Electromyographic activity of plantar flexors and dorsiflexors was recorded prior to H-reflex stimulation ( $-500\text{--}0\text{ ms}$ , Interval 1). Dorsiflexion torque was assessed prior to (Interval 1) and following ( $100\text{--}600\text{ ms}$ , Interval 2) H-reflex stimulation.

interneuron prevents excitation of the antagonistic alpha motor neuron pool and diminishes antagonistic muscle contraction. Without *DRI*, both muscle groups would contract simultaneously (Crone & Nielsen, 1994), leading to poor intermuscular coordination.

In the context of functional neuromechanics, reciprocal inhibition is a ubiquitous phenomenon which is considered to be of major relevance in movement control (Crone, 1993). Increased reciprocal inhibition in appropriate muscle groups has been shown to increase strength and flexibility (Blazevich et al., 2012; Geertsen, Lundbye-Jensen, & Nielsen, 2008; Nielsen & Kagamihara, 1992), improve performance in fine motor tasks that require a high degree of accuracy (Floeter, Danielian, & Kim, 2013), prevent injury (Shrier, 2007), and diminish muscle spasms in patients suffering from neurological disorders (Morita, Crone, Christenhuis, Petersen, & Nielsen, 2001). Beyond the functional aspects, reciprocal inhibition is a key mechanism by which to regulate the level of antagonistic co-contraction (Geertsen et al., 2008); thus, agonistic muscle contraction (initiating a movement) reduces the tension in the antagonistic muscle (opposing the movement), which simultaneously relaxes (Crone & Nielsen, 1994). As a consequence, motor coordination becomes more efficient, accurate, and economical (Floeter et al., 2013; Lavoie, Devanne, & Capaday, 1997; Nielsen & Kagamihara, 1992).

Anecdotal evidence indicates potential modulation of *DRI* by WBV; however, no firm evidence regarding the mechanism exists. Nevertheless, experiments have indicated a persistent vibration-induced modulation at the spinal and supraspinal level. Reduced Ia afferent transmission (Krause, Gollhofer, Freyler, Jablonka, & Ritzmann, 2016; Sayenko, Masani, Alizadeh-Meghraz, Popovic, & Craven, 2010) occurs concomitantly with facilitation of supraspinal pathways projecting onto the alpha motoneuron pool (Krause et al., 2016; Mileva, Bowtell, & Kossev, 2009). Particularly notable effects have been demonstrated for WBV exercises executed at a frequency of 30 Hz (Krause et al., 2016) and an amplitude of 4 mm (Ritzmann, Kramer, Gollhofer, & Taube, 2013). Furthermore, local vibration applied to the muscle belly indicates increased corticospinal excitability in the vibrated muscle (Rosenkranz & Rothwell, 2003), while the non-vibrated antagonistic muscle is simultaneously suppressed (Liepert & Binder, 2010; Rosenkranz & Rothwell, 2003). Cody, Henley, Parker, and Turner (1998) found an increase in *DRI* during local vibration treatment applied to hand musculature. None of these studies, however, applied H-reflex conditioning techniques to clearly distinguish the contribution of *DRI* to WBV-induced benefits in movement control (Crone et al., 1994; Geertsen et al., 2008).

Taken together, valuable preliminary findings and limited current knowledge persuaded us to carry out an experiment aimed at evaluating the immediate effect of WBV on *DRI* and antagonistic co-contraction of muscles encompassing the ankle joint (Pel et al.,



**Fig. 2. Experimental protocol.** Eight submaximal isometric contractions at 30% MVC were executed before and after a 1-min session of WBV in each set. Sets were repeated five times and separated by a 15-min rest period.

2009; Pollock, Woledge, Mills, Martin, & Di Newham, 2010; Ritzmann, Kramer et al., 2013). Based on biomechanical and neurophysiological methodologies, we expect this experiment to increase the understanding of the control and organization of human movement after WBV. We hypothesized that WBV would increase *DRI* during submaximal isometric dorsiflexion, and that increased *DRI* would be associated with reduced co-contraction of antagonistic plantar flexors.

## 2. Materials and methods

### 2.1. Experimental design

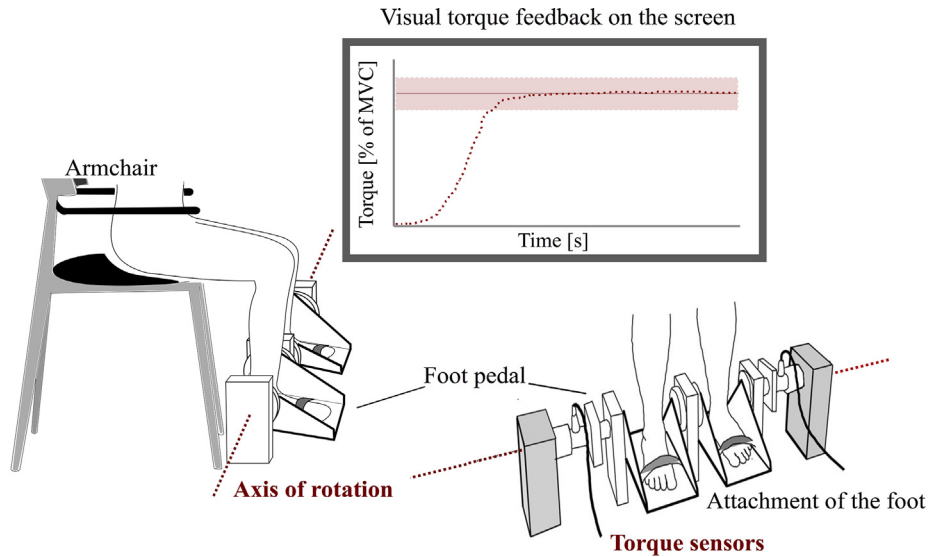
A single-group repeated-measures study design was used to evaluate the acute effect of WBV on *DRI*, neuromuscular activity, and antagonistic co-contraction from ankle plantar flexors to ankle dorsiflexors during submaximal isometric dorsiflexion. This paradigm was chosen in order to establish modulation in a functionally relevant task in accordance with Lundbye-Jensen and Nielsen (2008) and Geertsen et al. (2008). We selected submaximal isometric dorsiflexion due to its excellent reproducibility (Webber & Porter, 2010) and measurability of *DRI* (Geertsen et al., 2008). All measurements were executed prior to and immediately after a 1-min session of WBV, in time intervals of three minutes (Fig. 2). Measurements were executed in five sets including  $2 \times 8$  repetitions; sets were separated by breaks of 15 min to avoid fatigue and to ensure that the H-reflex recovered to baseline (Krause et al., 2016).

### 2.2. Participants

Eighteen subjects (eleven females, seven males, age  $23 \pm 3$  years old, body mass  $69 \pm 5$  kg; height  $171 \pm 7$  cm, values expressed as the mean  $\pm$  standard deviation) volunteered to participate in the present study. All subjects gave written informed consent for the experimental procedure, which was approved by the Ethics Committee of the University of Freiburg (197/17) and conducted in full accordance with the latest version of the Declaration of Helsinki. The subjects were healthy with no previous neurological irregularities or injuries of the lower extremities. The sample size was estimated by means of a power analysis based on a previously executed pilot study ( $f = 0.90$ ;  $\alpha = 0.05$ ; power = 0.90).

### 2.3. WBV training procedure

The WBV device was a side-alternating vibration platform (Galileo Sport, Novotec Medical, Pforzheim, Germany) that generates oscillations along the sagittal axis to the human body standing on the vibration platform. In the present study, the axis of rotation was placed in between the subjects' feet, which were placed 21 cm away from the axis of rotation, resulting in a vibration amplitude of



**Fig. 3. Armchair and force recordings.** Illustration of the armchair and foot pedal used to record submaximal isometric dorsiflexion. The rotation axis of the upper ankle joint coincided with the rotation axis of the foot pedal. The subjects were sitting in the armchair, with the ankle, knee, and hip joints flexed at 90°. The subjects received a visual torque feedback on a screen. The dotted line illustrates the actual torque and the red bar illustrates the acceptable boundaries of the individual's torque (30% MVC  $\pm$  5% MVC) to achieve a valid attempt. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

4 mm. During the 1-min WBV session, the vibration frequency was set to 30 Hz with a peak acceleration of 7.2 g (Abercromby et al., 2007). Subjects maintained a static body position with a knee angle of 10° and a forefoot stance (Ritzmann, Gollhofer, & Kramer, 2013). This parameter selection was justified by recent studies that investigated the effects of WBV on the H-reflex (Krause et al., 2016; Ritzmann, Kramer et al., 2013). The intermittent protocol with a repetitive 60-s WBV exposure followed by a break was chosen with reference to Cochrane (2011), who identified this exercise mode as one of the most common.

Subjects were instructed to place their hands on their hips, direct their head and eyes forward, and distribute their weight equally between both feet.

#### 2.4. Testing procedure

Subjects were comfortably seated in an armchair (Fig. 3) located above a force pedal, and the right leg was positioned with the hip, knee, and ankle joint flexed at 90° (Billot, Simoneau, Ballay, van Hoecke, & Martin, 2011). The right foot was firmly attached to the force pedal with adjustable straps. Prior to the experimental session, subjects performed a warm-up including brief isometric dorsiflexion movements (1 s) to approximately half the maximal voluntary contraction (MVC) torque (3 s) followed by three isometric dorsiflexion movements to MVC (4 s). The highest torque [Nm] value of three MVCs served as a reference for the subsequent experiment (Geertsen et al., 2008).

The experimental sessions involved submaximal isometric dorsiflexion movements at 30% of MVC before and after WBV, to provide a high repeatability without fatigue (Aniss, Gandevia, & Burke, 1992). All participants were instructed to keep a constant body position with the head straight and refrain from talking while measurements were taken. Visual feedback of the torque applied to the pedal was given by a moving graph monitored on the screen in front of the subject. First, an auditory signal was given to alert the subjects to be ready, followed by a second signal to begin the submaximal isometric dorsiflexion movements with the aim of keeping the torque on a constant plateau. Dorsiflexion torque was held until the third signal (delayed by 4 s) (Fig. 1C). This paradigm was repeated 16 times in one set; eight times prior to WBV and eight times after. Repetitions were separated by 15-s pauses to avoid post-activation depression (Grey et al., 2008).

#### 2.5. Outcome measures

##### 2.5.1. Force recordings

Maximal isometric dorsiflexion torque [Nm] was established prior to the experimental session (Table 1). During the experimental session, ankle joint torques were recorded during isometric dorsiflexion at 30% of the maximal torque hold for 3 s (Fig. 1). Trials were excluded in the case of a countermovement (defined as a downward deflection of the baseline), an overshoot or undershoot (defined as a dorsiflexion torque > 35%/ < 25% of MVC torque between 0 and 100 ms after H-reflex stimulation, respectively). There was never more than one trial excluded in a session.

**Table 1**

Results of the H/M recruitment curves.

<i>Before and after the experimental session</i> H <sub>max</sub> /M <sub>max</sub> ratios	<i>Before experiment</i>	<i>After experiment</i>	<i>Statistics</i> Cronbach's $\alpha$	<i>ES</i>
M. tibialis anterior	0.20 ± 0.1	0.20 ± 0.1	0.977	≈
M. soleus	0.66 ± 0.2	0.65 ± 0.2	0.773	≈
M. gastrocnemius medialis	0.27 ± 0.2	0.27 ± 0.2	0.971	
<i>Experimental session–kinematic</i> Ankle joint torque [Nm]	<i>Pre-WBV</i>	<i>Post-WBV</i>	<i>Statistics</i> Cronbach's $\alpha$	<i>ES</i>
I <sub>1</sub> before stimulation	9.92 ± 2.2	9.97 ± 2.1	0.946	≈
I <sub>2</sub> after the stimulation	9.81 ± 2.2	9.91 ± 2.2	0.948	≈
Joint position [°]				
Hip joint position	90.4 ± 4.7	89.5 ± 5.6	0.915	≈
Knee joint position	90.1 ± 4.4	90.1 ± 5.1	0.887	≈
Ankle joint position	89.9 ± 4.7	89.5 ± 4.8	0.893	≈
<i>Experimental session –neuromuscular</i> EMG [mVs]	<i>Pre-WBV</i>	<i>Post-WBV</i>	<i>Statistics</i> ANOVA ( <i>p</i> , <i>F</i> )	<i>Cohen's d</i>
M. tibialis anterior	33.8 ± 13.3	32.1 ± 12.8	<i>p</i> = 0.009, <i>F</i> = 8.768*	0.600
M. soleus	6.6 ± 4.3	6.5 ± 4.1	<i>p</i> = 0.002, <i>F</i> = 13.177*	0.827
M. gastrocnemius medialis	10.6 ± 5.8	10.5 ± 5.9	<i>p</i> = 0.592, <i>F</i> = 0.298	0.159
Co-contraction of antagonistic muscles				
M. tibialis anterior/M. soleus			<i>p</i> = 0.054, <i>F</i> = 4.301	0.742

The **top** displays the grand means of the H<sub>max</sub>/M<sub>max</sub> ratios as calculated from the H/M recruitment curves obtained prior to and following the experimental session. The **middle** and **bottom** display the grand means obtained for the key kinematic and neuromuscular parameters, respectively, recorded during the experimental session: the mean isometric voluntary dorsiflexion torque assessed for the I<sub>1</sub> (500 ms prior to H-reflex stimulation) and I<sub>2</sub> (100–600 ms following H-reflex stimulation) periods; the average of the ankle, knee, and hip joint positions for the period 500 ms before to 600 ms after H-reflex stimulation; the mean EMG activity for the period 500 ms before to stimulation onset; and antagonistic co-contraction of the target muscles m. tibialis anterior and m. soleus (TA/SOL).

Parameters marked with the symbol ≈ are statistically equal; parameters marked with the symbol \* differed significantly. ES = equivalence statistics; I = time interval.

### 2.5.2. Electromyographic (EMG) recordings

Bipolar Ag/AgCl surface electrodes (Ambu Blue Sensor P, Ballerup, Denmark; diameter 9 mm, center-to-center distance 34 mm) were placed over the musculus soleus (SOL), the musculus gastrocnemius medialis (GM), and the musculus tibialis anterior (TA) of the right leg according to SENIAM (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). The longitudinal axes of the electrodes were in line with the direction of the underlying muscle fibers. The reference electrode was placed on the patella. Interelectrode resistance was kept below 2 kΩ by means of shaving, light abrasion, degreasing, and disinfection of the skin. The EMG signals were transmitted to the amplifier (band-pass filter 10 Hz–1 kHz, 1000x amplification) via shielded cables, and recorded with 4 kHz. The cables were carefully taped to the skin.

### 2.5.3. H-reflex stimulation

SOL H-reflexes were elicited through peripheral nerve stimulation (PNS) with single rectangular pulses of 1 ms (Digitimer DS7, Digitimer, Welwyn Garden City, UK). The anode (10 cm × 5 cm dispersal pad) was fixed directly below the patella on the anterior aspect of the knee. The cathode (2 cm in diameter) was placed in the popliteal fossa and moved until the best position was found to elicit an H-reflex in the SOL by depolarizing the posterior tibial nerve. H/M recruitment curves for SOL and GM were recorded before and after the experimental session (Crone, Hultborn, Jespersen, & Nielsen, 1987; Crone & Nielsen, 1989; Crone et al., 1994). During the experimental session, the stimulation current was set to elicit an H-reflex with an amplitude of 15–25% of maximal muscular responses (M<sub>max</sub>) for all measurements (Crone et al., 1990).

### 2.5.4. H-reflex conditioning for evaluation of DRI

DRI of the SOL H-reflex was evoked by conditioning stimulation of the common peroneal nerve (CPN) innervating the TA through bipolar surface electrodes (diameter 0.5 cm; Blue Sensor, Ambu) placed 2–3 cm distal to the neck of the fibula, according to Lundbye-Jensen and Nielsen (2008) (Fig. 1A). Great care was taken to ensure that the conditioning stimulus was applied at a location where the threshold for an M response (motor threshold (MT)) in the TA was lower than the MT in the peroneal muscle (Geertsen et al., 2008). An H/M recruitment curve was recorded prior to and following the experimental session to check for reliability. In all subjects, a time course of the effect of CPN stimulation on the SOL H-reflex was obtained at rest. A stimulation strength of 1.1x MT was employed in all trials to obtain a small M response in the TA muscle, which could be monitored throughout the experiment and used

to ensure that the effect of the conditioning stimulus was comparable in all trials (Petersen, Morita, & Nielsen, 1998). Higher stimulation intensities were avoided to minimize the influence of pathways differing from disynaptic Ia reciprocal pathways on the inhibition (Petersen et al., 1998). A submaximal stimulation intensity of 1.1x MT did not activate all inhibitory interneurons, permitting an assessment of potential facilitatory and inhibitory effects of WBV (Petersen et al., 1998).

An H-reflex was elicited every 15 s; and either occurred in isolation (control, H-reflex<sub>CON</sub>) or was preceded by a conditioning stimulation (H-reflex<sub>CONDITIONED</sub>) of the CPN with conditioning-test (CT) intervals from 1 to 6 ms in 1-ms steps. Ten control and ten conditioned reflexes were sampled at each of the six CT intervals (Geertsens et al., 2008). The CT interval that caused the largest inhibition (2-ms CT interval: #5 subjects, 3-ms CT interval: #12 subjects, 4-ms CT interval: #1 subject) was used to assess *DRI* in the experimental session (Fig. 1B and C). During these intervals, it is likely that only *DRI* contributed to the measured inhibition (Crone et al., 1987). Stimulation was triggered 1 s after reaching the 30% MVC plateau (approximately 2 s after the beginning of contraction and 1 s before the end of contraction) (Fig. 1B). Trials with H-reflex<sub>CON</sub> and H-reflex<sub>CONDITIONED</sub> were randomized. This paradigm was repeated until eight trials of each condition were obtained before and after WBV. Each individual response was measured and the average SOL H-reflex amplitudes were calculated for each condition. The size of the control H-reflex was adjusted to 15–25% of  $M_{\max}$  for all conditions (Crone et al., 1990).

### 2.5.5. Joint goniometry

Ankle (dorsiflexion and plantar flexion), knee (flexion and extension), and hip (flexion and extension) joint kinematics in the sagittal plane were recorded using electrogoniometers (Biometrics®, Gwent, UK), which were fixed at the respective joints according to Ritzmann, Gollhofer et al. (2013). All signals were recorded with a sampling frequency of 1 kHz.

## 2.6. Data processing

For the H/M recruitment curves, peak-to-peak amplitudes of the H-reflexes and M-waves were calculated [mV] and plotted against the stimulation current.  $H_{\max}$  and  $M_{\max}$  were assessed, and  $H_{\max}/M_{\max}$ -ratios were calculated. For *DRI*, the peak-to-peak amplitudes of SOL H-reflex<sub>CONDITIONED</sub> responses were expressed relative to the obtained H-reflex<sub>CON</sub> amplitudes.

The EMG activities of dorsiflexors and plantarflexors (TA, SOL, and GM) were analyzed for a period of 500 ms immediately prior to H-reflex stimulation (Interval 1 =  $I_1$ ). The integrals were calculated from the rectified raw EMG signals, normalized to the corresponding  $M_{\max}$ , and time normalized [mVs].

To investigate the degree of co-contraction during dorsiflexion, antagonistic muscle activity (SOL) was expressed in relation to agonistic muscle activity (TA) during a time frame of 500 ms immediately prior to H-reflex stimulation ( $I_1$ ).

The mean voluntary dorsiflexion torque [Nm] was assessed for a period of 500 ms immediately prior to H-reflex stimulation ( $I_1$ ), in addition to 100–600 ms immediately following the stimulation (Interval 2 =  $I_2$ ; Fig. 1C). Values from all the trials before and after WBV were averaged for each subject.

Mean ankle, knee, and hip joint positions (°) were calculated for the 500-ms period prior to  $I_1$  to the 600-ms period following stimulation ( $I_2$ ). Values from all the trials before and after WBV were averaged for each subject.

## 2.7. Statistics

Statistical analyses were executed using SPSS 23.0 (SPSS Inc., Chicago, Illinois). The effects of WBV on the variables *DRI* and the EMG activities of TA, SOL, and GM, in addition to SOL/TA co-contraction were evaluated using a one-factor analysis of variance (ANOVA); time [2, pre vs. post]. *A priori*, the normality of the data was evaluated using the Kolmogorov–Smirnov test, which indicated that the data followed a normal distribution. If the assumption of sphericity, as measured by Mauchly's test, was violated, Greenhouse–Geisser correction was used. To correct for multiple testing, the false discovery rate was controlled according to the Benjamini–Hochberg–Yekutieli method, which conceptualizes the rate of type I errors (Benjamini & Hochberg, 1995; Benjamini & Yekutieli, 2005). Effect sizes were calculated using Cohen's *d*.

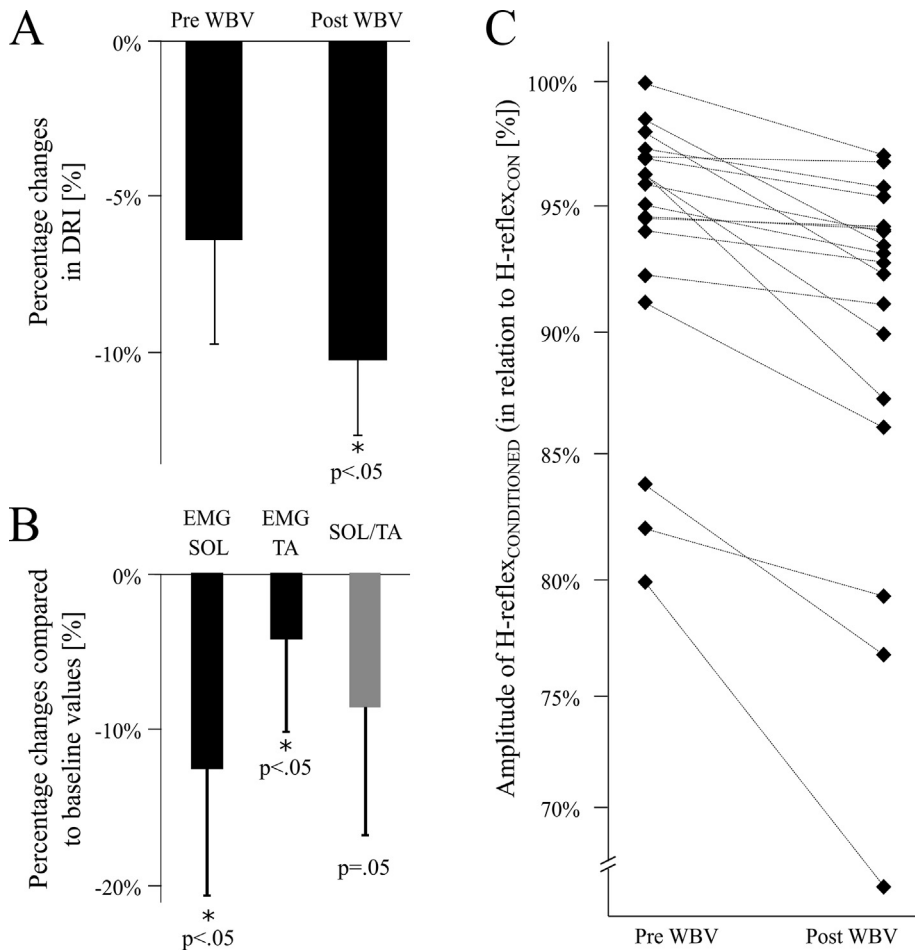
Bivariate, two-tailed Pearson correlation analyses were conducted to determine the strength of the linear relationship between the WBV-induced change in *DRI* and the SOL/TA ratio.

To ensure that particular variables (ankle joint torque,  $H_{\max}/M_{\max}$  ratio, and body position assessed by goniometric recordings of the ankle and knee joint) did not change over time in response to WBV, Cronbach's  $\alpha$  values (Fleiss, 1986) and equivalence statistics (ES), (Borman, Chatfield, Damjanov, & Jackson, 2009) were calculated. To test for statistical equivalence, the 95% confidence intervals were calculated for the differences between pre- and post-WBV. The acceptable bounds were determined based on Ritzmann, Kramer et al. (2013) using repeated measurement without intervention (Borman et al., 2009). If the confidence interval stayed within these bounds, the differences were statistically equal. In the case of statistical equivalence, the respective parameter is marked with a symbol ( $\approx$ ). Group data are presented as the mean  $\pm$  standard deviation (SD).

## 3. Results

### 3.1. H-reflexes

$H_{\max}/M_{\max}$ -ratios obtained from the H/M recruitment curves for SOL and TA (SOL  $-0.6 \pm 7.6\%$ , TA  $+3.2 \pm 20.0\%$ , GM  $+4.4 \pm 28.5\%$ ) after the experimental session were statistically equal to those measured prior to the WBV. Cronbach's  $\alpha$  estimates



**Fig. 4.** Changes in disynaptic reciprocal inhibition (*DRI*) following whole-body vibration. Grand means of disynaptic reciprocal inhibition (*DRI*) illustrate a significant increase during submaximal isometric dorsiflexion following a 1-min session of WBV as compared with prior (A). Concomitantly, electromyographic activities in the musculus soleus (SOL) and musculus tibialis anterior (TA), and the SOL/TA ratio after indicate that antagonistic co-contraction was slightly reduced after WBV (B). C illustrates the WBV-induced changes in *DRI* for each participant as calculated by the  $H\text{-reflex}_{\text{CONDITIONED}}/H\text{-reflex}_{\text{CON}}$  ratio.

yielded excellent values for SOL, TA, and GM (Table 1).

### 3.2. Joint torques

Grand means of the dorsiflexion torques are illustrated in Table 1; and data of one representative subject illustrating a 30% MVC contraction are displayed in Fig. 1B. Cronbach's  $\alpha$  estimates yielded excellent values (Table 1). *ES* revealed statistical equivalence for ankle joint torques obtained before ( $29.3 \pm 1.6\%$ ,  $29.0 \pm 0.21\%$  of the maximal peak torque) and after WBV ( $29.5 \pm 1.9\%$ ,  $29.2 \pm 1.9\%$ ), indicating that the force profiles were kept constant between the measurements.

### 3.3. Disynaptic reciprocal inhibition (*DRI*)

A significantly increased *DRI* from TA to SOL was revealed after WBV (pre-WBV  $-6.3 \pm 2.4\%$ , post-WBV  $-10.1 \pm 3.3\%$ , Cohen's  $d = 0.807$ ). Fig. 4A illustrates the grand means of *DRI* expressed as percentage changes in the  $H\text{-reflex}_{\text{CONDITIONED}}/H\text{-reflex}_{\text{CON}}$  ratio prior to and following WBV. Fig. 4C illustrates the WBV-induced changes in *DRI* for each participant.

### 3.4. Electromyography (*EMG*)

Background EMGs are illustrated in Table 1 and Fig. 4B. GM revealed no WBV-induced changes (GM  $+3.1 \pm 25.6\%$ ). TA EMG activity was significantly reduced by  $4.3 \pm 5.9\%$ , and SOL EMG was reduced by  $12.6 \pm 16.2\%$ .

### 3.5. Co-contraction – SOL/TA ratio

Antagonistic co-contraction (Fig. 4B) showed a strong tendency to be reduced following WBV ( $p = 0.05$ ,  $-8.2\%$ , Table 1).

### 3.6. Joint position

Joint positions are displayed in Table 1. Cronbach's  $\alpha$  estimates yielded excellent values for the hip joint and good values for the ankle and knee joints (Table 1). ES revealed statistical equivalence for angular excursions at the ankle ( $-0.4 \pm 3.3\%$ ), knee ( $+0.0 \pm 3.4\%$ ), and hip ( $-0.8 \pm 5.3\%$ ) joints, indicating that the body position was kept constant between the measurements.

### 3.7. Correlations

A significant negative correlation was detected for the variables *DRI* and the SOL/TA ratio ( $r = -0.86$ ,  $p = 0.007$ ).

## 4. Discussion

The present study aimed to evaluate the effect of WBV on *DRI* and antagonistic co-contraction from ankle plantar flexors to ankle dorsiflexors during submaximal isometric dorsiflexion. The main finding was that *DRI* was facilitated during submaximal dorsiflexion. Coincidentally, neuromuscular activation of TA and SOL was reduced and the SOL/TA ratio tended toward a diminished antagonistic co-contraction, whereas the dorsiflexion torques remained unchanged.

### 4.1. WBV and *DRI*

The WBV-induced inhibition of spinal reflexes, which has been established as a highly diminished Ia afferent transmission, indicates a strong effect of mechanical oscillation on afferent feedback integration at the alpha motoneuron pool (Krause et al., 2016; Sayenko et al., 2010). The SOL H-reflex reduction in response to WBV has been reported to occur within a percentage range of 19–49%, and depending on the duration of WBV application (Leonard, Moritani, Hirschfeld, & Forssberg, 1990), presents a persistence varying from 1 (Sayenko et al., 2010) to 10 min (Krause et al., 2016) following the WBV treatment. With an emphasis on the shank muscles, the findings of the current study suggest that *DRI* is a mechanism involved in the regulation of feedback inhibition at the spinal level. In accordance with the decrease in muscle co-activation, the results further indicate that an increase in *DRI* is associated with an improved antagonistic muscle coordination and economization of mono-articular movement involving muscle groups that are instantly affected by the vibration stimulus due to their proximity to the vibration device (Pel et al., 2009; Pollock et al., 2010; Ritzmann, Gollhofer et al., 2013).

### 4.2. Antagonistic co-activation after WBV and its relevance for movement coordination

The simultaneous decline in SOL and TA EMG activities indicates an economization of isometric contraction; the EMG activities of the muscles are related to the extent of fiber recruitment (Aagaard, 2003; Milner-Brown, Stein, & Yemm, 1973a, 1973b) and frequency (Aagaard, 2003; Milner-Brown et al., 1973a). The lower EMG activity following WBV is a result of a smaller number of recruited muscle fibers and diminished motor unit discharge frequencies (Moritani & Muro, 1987), although dorsiflexion torque generated by the target muscle remained identical (Freund, Budingen, & Dietz, 1975; Moritani & Muro, 1987). We conclude that the intermuscular coordination of antagonistic co-contraction, most likely caused by an increased *DRI*, may have been the key mechanism underlying the WBV-induced changes. *DRI* achieved simultaneous relaxation of SOL by the contraction of its agonistic TA (Crone & Nielsen, 1994) (Table 1), although the antagonistic SOL showed only a minor activation during the dorsiflexion contraction prior to WBV (Fig. 2). The strong tendency of a reduced co-contraction supported by great effect sizes is in accordance with these observations, and supports a conclusive statement disregarding the experimental paradigm; usually, there is less risk of stretching the antagonist as compared with a dynamic contraction during isometric contractions (Geertsens et al., 2008).

### 4.3. Functional significance and prospect

The theoretical implications of enhanced reciprocal inhibition may be of great relevance during everyday movements. Each joint is controlled by two opposing sets of muscles, extensors and flexors, which must work in synchrony. Reciprocal inhibition reduces the co-contraction of antagonists encompassing a joint (Lavoie et al., 1997; Nielsen & Kagamihara, 1992) by reducing activation intensity, and thus, the counter-forces opposing the movement trajectory generated by antagonists. Therefore, reciprocal inhibition allows the central nervous system to control joint torques or positioning more accurately (Nielsen & Kagamihara, 1992), facilitating ease of movement (Geertsens et al., 2008; Nielsen & Kagamihara, 1992); and is considered to be a safeguard against injury (Shrier, 2007). Based on the present experimental findings, we conclude that WBV may be helpful in promoting a high level of movement control, which can be advantageous in a number of ways. *Firstly*, healthy individuals can benefit from a session of WBV followed by a task for which the level of *DRI* is relevant. For instance, in the stretch-shortening cycle (Taube, Leukel, & Gollhofer, 2012) or perturbed posture control (Grey, Ladouceur, Andersen, Nielsen, & Sinkjaer, 2001), the risk of SOL stretch reflex elicitation may be reduced at the onset of rapid ankle flexion. For both movement modalities, WBV has been shown to be advantageous and cause



performance improvements (for review see [Rittweger, 2010](#)). A *second* aspect deals with the application of WBV in specific sub-populations suffering from movement disorders and spasticity ([Crone, Petersen, Nielsen, Hansen, & Nielsen, 2004](#); [Knikou & Mummidisetty, 2011](#); [Morita et al., 2001](#); [Okuma & Lee, 1996](#)). In patients with neurological disorders, impairment or degeneration of the CNS (stroke, cerebral or hemiparetic palsy, multiple sclerosis) is mostly accompanied by reduced reciprocal inhibition and an increased antagonistic co-activation associated with uneconomical and inaccurate movements ([Boorman, Lee, Becker, & Windhorst, 1996](#); [Cheng, Ju, Chen, Chuang, & Cheng, 2015](#); [Crone et al., 2004](#); [Liepert & Binder, 2010](#)). Experimental evidence suggests that spastic individuals are unable to perform rapid movements with a full angular range of motion due to co-activation of the antagonistic muscle ([Boorman et al., 1996](#); [Crone et al., 1994](#); [Leonard et al., 1990](#)) resulting from exaggerated muscle spindle reflex sensitivity and pathologically reduced ability to increase reciprocal inhibition ([Crone et al., 1994](#); [Xia & Rymer, 2005](#)). WBV, in addition to local vibration, have been proven to improve movement accuracy ([Cheng, Yu, Wong, Tsai, & Ju, 2015](#); [Haas, Turbanski, Kessler, & Schmidtbleicher, 2006](#)), increase the active range of motion ([Cheng et al., 2015](#); [Krause et al., 2017](#)), and significantly reduce co-contraction of antagonists ([Krause et al., 2017](#)). Supported by the neuromechanical consequences related to an increased *DRI* provided in the current study, we suggest that WBV may be an appropriate therapy in the clinical treatment of spasticity-related side effects of neurological disorders, smoothing and ameliorating voluntary movement control in everyday life.

#### 4.4. Study limitations

For a conclusive statement, it is crucial to consider the limitations of the present study. Two aspects are of importance; the first deals with the study protocol, and the second with the methodological approach. Although the present study provides scientific evidence for an increase in *DRI* following a 1-min session of acute WBV, the findings are restricted to the selected vibration parameters; 30 Hz, 4 mm amplitude, and 7.2 g peak acceleration ([Abercromby et al. \(2007\)](#) and [Ritzmann, Gollhofer et al. \(2013\)](#)). We are therefore uncertain whether our findings are applicable to the various WBV exercise protocols or settings with differing WBV durations, breaks, or WBV types (synchronous or stochastic vibration; [Rittweger, 2010](#)). As a test paradigm to assess vibration-induced changes in *DRI*, we used submaximal MVCs; thus, the transferability to other types of contraction, including maximal MVCs ([Lundbye-Jensen and Nielsen \(2008\)](#)) or rate of force development ([Geertsen et al., 2008](#)), is uncertain and needs to be addressed in future experiments.

#### 5. Conclusions

An increased *DRI* concomitant with reduced antagonistic and agonistic muscle activation, which tends to result in a diminished co-contraction while the resulting joint torque remains constant, indicates an economization of movement following WBV treatment. In a functional context, when an agonistic muscle contracts, to cause the desired motion, it is useful to force the antagonists to relax in order to diminish the counterforces directed against the desired movement direction. Thus, we conclude that WBV, with the benefits of easy application and passive training, may be helpful to execute accurate motion with remarkably high quality.

Beyond *DRI*, additional mechanisms such as presynaptic inhibition ([Gillies, Lance, Neilson, & Tassinari, 1969](#)), post-activation depression ([Kipp, Johnson, Doeringer, & Hoffman, 2011](#)), and intracortical modulation ([Mileva et al., 2009](#); [Rosenkranz & Rothwell, 2003](#)) have been discussed in vibration-associated investigations. With an emphasis on functional significance, further evidence based on neurophysiological methodologies is needed to make a conclusive statement regarding the central and peripheral circuitries involved in neuromuscular modulation in response to WBV.

#### Conflicts of interest

The authors have no conflicts of interest to declare.

#### Disclosure

There are no financial or other conflicts of interest associated with this work.

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#### Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.humov.2018.06.011>.

#### References

- Aagaard, P. (2003). Training-induced changes in neural function. *Exercise and Sport Sciences Reviews*, 31, 61–67.
- Abercromby, A. F. J., Amonette, W. E., Layne, C. S., McFarlin, B. K., Hinman, M. R., & Paloski, W. H. (2007). Variation in neuromuscular responses during acute whole-

- body vibration exercise. *Medicine and Science in Sports and Exercise*, 39, 1642–1650.
- Aniss, A. M., Gandevia, S. C., & Burke, D. (1992). Reflex responses in active muscles elicited by stimulation of low-threshold afferents from the human foot. *Journal of Neurophysiology*, 67, 1375–1384.
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the False Discovery Rate: A Practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society. Series B (Methodological)*, 57, 289–300.
- Benjamini, Y., & Yekutieli, D. (2005). False discovery rate-adjusted multiple confidence intervals for selected parameters. *Journal of American Statistical Association*, 100, 71–81.
- Billot, M., Simoneau, E. M., Ballay, Y., van Hoesck, J., & Martin, A. (2011). How the ankle joint angle alters the antagonist and agonist torques during maximal efforts in dorsi- and plantar flexion. *Scandinavian Journal of Medicine and Science in Sports*, 21, e273–e281.
- Blazevich, A. J., Kay, A. D., Waugh, C., Fath, F., Miller, S., & Cannavan, D. (2012). Plantarflexor stretch training increases reciprocal inhibition measured during voluntary dorsiflexion. *Journal of Neurophysiology*, 107, 250–256.
- Boorman, G. I., Lee, R. G., Becker, W. J., & Windhorst, U. R. (1996). Impaired “natural reciprocal inhibition” in patients with spasticity due to incomplete spinal cord injury. *Electroencephalography and Clinical Neurophysiology*, 101, 84–92.
- Borman, P. J., Chatfield, M. J., Damjanov, I., & Jackson, P. (2009). Design and analysis of method equivalence studies. *Analytical Chemistry*, 81, 9849–9857.
- Cheng, H.-Y. K., Ju, Y.-Y., Chen, C.-L., Chuang, L.-L., & Cheng, C.-H. (2015). Effects of whole-body vibration on spasticity and lower extremity function in children with cerebral palsy. *Human Movement Science*, 39, 65–72.
- Cheng, H.-Y. K., Yu, Y.-C., Wong, A. M.-K., Tsai, Y.-S., & Ju, Y.-Y. (2015). Effects of an eight-week whole-body vibration on lower extremity muscle tone and function in children with cerebral palsy. *Research in Developmental Disabilities*, 38, 256–261.
- Cochrane, D. J. (2010). The potential neural mechanisms of acute indirect vibration. *Journal of Sports Science & Medicine*, 10, 19–30.
- Cochrane, D. J. (2011). Vibration exercise: The potential benefits. *International Journal of Sports Medicine*, 32, 75–99.
- Cochrane, D. J., Stannard, S. R., Firth, E. C., & Rittweger, J. (2010). Acute whole-body vibration elicits post-activation potentiation. *European Journal of Applied Physiology*, 108, 311–319.
- Cody, F. W., Henley, N. C., Parker, L., & Turner, G. (1998). Phasic and tonic reflexes evoked in human antagonistic wrist muscles by tendon vibration. *Electroencephalography and Clinical Neurophysiology*, 109, 24–35.
- Crone, C. (1993). Reciprocal inhibition in man. *Danish Medical Bulletin*, 40, 571–581.
- Crone, C., Nielsen, J., Petersen, N., Ballegaard, M., & Hultborn, H. (1994). Disynaptic reciprocal inhibition of ankle extensors in spastic patients. *Brain: A Journal of Neurology*, 117, 1161–1168.
- Crone, C., Petersen, N. T., Nielsen, J. E., Hansen, N. L., & Nielsen, J. B. (2004). Reciprocal inhibition and corticospinal transmission in the arm and leg in patients with autosomal dominant pure spastic paraparesis (ADPSP). *Brain: A Journal of Neurology*, 127, 2693–2702.
- Crone, C., Hultborn, H., Jespersen, B., & Nielsen, J. (1987). Reciprocal Ia inhibition between ankle flexors and extensors in man. *The Journal of Physiology*, 389, 163–185.
- Crone, C., Hultborn, H., Mazieres, L., Morin, C., Nielsen, J., & Pierrot-Deseilligny, E. (1990). Sensitivity of monosynaptic test reflexes to facilitation and inhibition as a function of the test reflex size: A study in man and the cat. *Experimental Brain Research*, 81, 35–45.
- Crone, C., & Nielsen, J. (1989). Spinal mechanisms in man contributing to reciprocal inhibition during voluntary dorsiflexion of the foot. *The Journal of Physiology*, 416, 255–272.
- Crone, C., & Nielsen, J. (1994). Central control of disynaptic reciprocal inhibition in humans. *Acta Physiologica Scandinavica*, 152, 351–363.
- Delecluse, C., Roelants, M., & Verschueren, S. (2003). Strength increase after whole-body vibration compared with resistance training. *Medicine and Science in Sports and Exercise*, 35, 1033–1041.
- Fleiss, J. L. (1986). Analysis of data from multiclinic trials. *Controlled Clinical Trials*, 7, 267–275.
- Floeter, M. K., Danielian, L. E., & Kim, Y. K. (2013). Effects of motor skill learning on reciprocal inhibition. *Restorative Neurology and Neuroscience*, 31, 53–62.
- Freund, H. J., Budingem, H. J., & Dietz, V. (1975). Activity of single motor units from human forearm muscles during voluntary isometric contractions. *Journal of Neurophysiology*, 38, 933–946.
- Geertsens, S. S., Lundbye-Jensen, J., & Nielsen, J. B. (2008). Increased central facilitation of antagonist reciprocal inhibition at the onset of dorsiflexion following explosive strength training. *Journal of Applied Physiology*, 105, 915–922.
- Gillies, J. D., Lance, J. W., Neilson, P. D., & Tassinari, C. A. (1969). Presynaptic inhibition of the monosynaptic reflex by vibration. *The Journal of Physiology*, 205, 329–339.
- Grey, M. J., Klinge, K., Crone, C., Lorentzen, J., Biering-Sorensen, F., Ravnborg, M., & Nielsen, J. B. (2008). Post-activation depression of soleus stretch reflexes in healthy and spastic humans. *Experimental Brain Research*, 185, 189–197.
- Grey, M. J., Ladouceur, M., Andersen, J. B., Nielsen, J. B., & Sinkjaer, T. (2001). Group II muscle afferents probably contribute to the medium latency soleus stretch reflex during walking in humans. *The Journal of Physiology*, 534, 925–933.
- Haas, C. T., Turbanski, S., Kessler, K., & Schmidtbleicher, D. (2006). The effects of random whole-body vibration on motor symptoms in Parkinson's disease. *NeuroRehabilitation*, 21, 29–36.
- Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology*, 10, 361–374.
- Kipp, K., Johnson, S. T., Doeringer, J. R., & Hoffman, M. A. (2011). Spinal reflex excitability and homosynaptic depression after a bout of whole-body vibration. *Muscle and Nerve*, 43, 259–262.
- Knikou, M., & Mummidisetty, C. K. (2011). Reduced reciprocal inhibition during assisted stepping in human spinal cord injury. *Experimental Neurology*, 231, 104–112.
- Krause, A., Gollhofer, A., Freyler, K., Jablonka, L., & Ritzmann, R. (2016). Acute corticospinal and spinal modulation after whole body vibration. *Journal of Musculoskeletal and Neuronal Interactions*, 16, 327–338.
- Krause, A., Schönau, E., Gollhofer, A., Duran, I., Ferrari-Malik, A., Freyler, K., & Ritzmann, R. (2017). Alleviation of motor impairments in patients with Cerebral Palsy: Acute effects of whole-body vibration on stretch reflex response, voluntary muscle activation and mobility. *Frontiers in Neurology*, 16, 416.
- Lavoie, B. A., Devanne, H., & Capaday, C. (1997). Differential control of reciprocal inhibition during walking versus postural and voluntary motor tasks in humans. *Journal of Neurophysiology*, 78, 429–438.
- Leonard, C. T., Moritani, T., Hirschfeld, H., & Forssberg, H. (1990). Deficits in reciprocal inhibition of children with cerebral palsy as revealed by H reflex testing. *Developmental Medicine and Child Neurology*, 32, 974–984.
- Liepert, J., & Binder, C. (2010). Vibration-induced effects in stroke patients with spastic hemiparesis: A pilot study. *Spinal Cord*, 28, 729–735.
- Lundbye-Jensen, J., & Nielsen, J. B. (2008). Immobilization induces changes in presynaptic control of group Ia afferents in healthy humans. *The Journal of Physiology*, 586, 4121–4135.
- Mileva, K. N., Bowtell, J. L., & Kossev, A. R. (2009). Effects of low-frequency whole-body vibration on motor-evoked potentials in healthy men. *Experimental Physiology*, 94, 103–116.
- Milner-Brown, H. S., Stein, R. B., & Yemm, R. (1973a). Changes in firing rate of human motor units during linearly changing voluntary contractions. *The Journal of Physiology*, 230, 371–390.
- Milner-Brown, H. S., Stein, R. B., & Yemm, R. (1973b). The orderly recruitment of human motor units during voluntary isometric contractions. *The Journal of Physiology*, 230, 359–370.
- Morita, H., Crone, C., Christenhuis, D., Petersen, N. T., & Nielsen, J. B. (2001). Modulation of presynaptic inhibition and disynaptic reciprocal Ia inhibition during voluntary movement in spasticity. *Brain: A Journal of Neurology*, 124, 826–837.
- Moritani, T., & Muro, M. (1987). Motor unit activity and surface electromyogram power spectrum during increasing force of contraction. *European Journal of Applied Physiology*, 56, 260–265.
- Ness, L. L., & Field-Fote, E. C. (2009). Effect of whole-body vibration on quadriceps spasticity in individuals with spastic hypertonia due to spinal cord injury.

- Restorative Neurology and Neuroscience, 27, 621–631.
- Nielsen, J. B. (2004). Sensorimotor integration at spinal level as a basis for muscle coordination during voluntary movement in humans. *Journal of Applied Physiology*, 96, 1961–1967.
- Nielsen, J., & Kagamihara, Y. (1992). The regulation of disynaptic reciprocal Ia inhibition during co-contraction of antagonistic muscles in man. *The Journal of Physiology*, 456, 373–391.
- Okuma, Y., & Lee, R. G. (1996). Reciprocal inhibition in hemiplegia: Correlation with clinical features and recovery. *The Canadian Journal of Neurological Sciences*, 23, 15–23.
- Pel, J. J. M., Bagheri, J., van Dam, L. M., van den Berg-Emons, H. J. G., Horemans, H. L. D., Stam, H. J., & van der Steen, J. (2009). Platform accelerations of three different whole-body vibration devices and the transmission of vertical vibrations to the lower limbs. *Medical Engineering & Physics*, 31, 937–944.
- Petersen, N., Morita, H., & Nielsen, J. (1998). Evaluation of reciprocal inhibition of the soleus H-reflex during tonic plantar flexion in man. *Journal of Neuroscience Methods*, 84, 1–8.
- Pierrot-Deseilligny, E., & Burke, D. C. (2012). *The circuitry of the human spinal cord: Its role in motor control and movement disorders* (4th ed.). Cambridge: Cambridge University Press.
- Pollock, R. D., Woledge, R. C., Mills, K. R., Martin, F. C., & Di Newham, J. (2010). Muscle activity and acceleration during whole body vibration: Effect of frequency and amplitude. *Clinical Biomechanics*, 25, 840–846.
- Rees, S. S., Murphy, A. J., & Watsford, M. L. (2008). Effects of whole-body vibration exercise on lower-extremity muscle strength and power in an older population: A randomized clinical trial. *Physical Therapy*, 88, 462–470.
- Rittweger, J. (2010). Vibration as an exercise modality: How it may work, and what its potential might be. *European Journal of Applied Physiology*, 108, 877–904.
- Ritzmann, R., Gollhofer, A., & Kramer, A. (2013b). The influence of vibration type, frequency, body position and additional load on the neuromuscular activity during whole body vibration. *European Journal of Applied Physiology*, 113, 1–11.
- Ritzmann, R., Kramer, A., Gollhofer, A., & Taube, W. (2013a). The effect of whole-body vibration on the H-reflex, the stretch reflex, and the short-latency response during hopping. *Scandinavian Journal of Medicine and Science in Sports*, 23, 331–339.
- Roelants, M., Delecluse, C., & Verschueren, S. M. (2004). Whole-body vibration training increases knee-extension strength and speed of movement in older women. *Journal of the American Geriatrics Society*, 52, 901–908.
- Rosenkranz, K., & Rothwell, J. C. (2003). Differential effect of muscle vibration on intracortical inhibitory circuits in humans. *The Journal of Physiology*, 551, 649–660.
- Sayenko, D. G., Masani, K., Alizadeh-Meghrizi, M., Popovic, M. R., & Craven, B. C. (2010). Acute effects of whole-body vibration during passive standing on soleus H-reflex in subjects with and without spinal cord injury. *Neuroscience Letters*, 482, 66–70.
- Shrier, I. (2007). Does stretching help prevent injuries? In D. MacAuley, & T. M. Best (Eds.). *Evidence-based medicine. Evidence-based sports medicine* (pp. 36–58). (2nd ed.). Malden, Mass, Oxford: BMJ Books/Blackwell Pub.
- Stark, C., Nikopoulou-Smyrni, P., Stabrey, A., Semler, O., & Schoenau, E. (2010). Effect of a new physiotherapy concept on bone mineral density, muscle force and gross motor function in children with bilateral cerebral palsy. *Journal of Musculoskeletal and Neuronal Interactions*, 10, 151–158.
- Taube, W., Leukel, C., & Gollhofer, A. (2012). How neurons make us jump: The neural control of stretch-shortening cycle movements. *Exercise and Sport Sciences Reviews*, 40, 106–115.
- Torvinen, S., Kannu, P., Sievänen, H., Järvinen, T. A. H., Pasanen, M., Kontulainen, S., ... Vuori, I. (2002). Effect of a vibration exposure on muscular performance and body balance. Randomized cross-over study. *Clinical Physiology and Functional Imaging*, 22, 145–152.
- Webber, S. C., & Porter, M. M. (2010). Reliability of ankle isometric, isotonic, and isokinetic strength and power testing in older women. *Physical Therapy*, 90, 1165–1175.
- Xia, R., & Rymer, W. Z. (2005). Reflex reciprocal facilitation of antagonist muscles in spinal cord injury. *Spinal Cord*, 43, 14–21.